What is sudden cardiac arrest?

Sudden cardiac arrest (SCA)is showed by a rapid "pulse" absence and an unconsciousness state caused by the inability of the heart to pump blood into the brain, and into the body, in an effective way. Usually sudden cardiac arrest is caused by potentially lethal arrhythmias and by some cardiac electric system abnormalities. It's defined "**sudden**" because of its nature, therefore it can affect any person in any place without any notice, even individuals who have never been previously diagnosed cardiac diseases or critic clinical conditions.
If sudden cardiac arrest is not treated immediately, in a few seconds, the person affected loses consciousness and for every minute passed without receiving any intervention, the percentage of surviving is reduced to 10 per cent. To save a patient's life affected with sudden cardiac arrest is necessary to proceed with a cardiopulmonary resuscitationand with a defibrillation to restore the cardiac rhythm before the brain is affected by the irreversible damage caused by the lack of blood and oxygen; these events occur between 4 and 6 minutes.

Do sudden cardiac arrest and cardiomyopathies have inherited components?

In developed countries, **sudden cardiac death** is responsible of more than **5% of total deaths** and of more of 50% of mortality for cardiovascular diseases. In Italy, it can be estimated, with a good approximation, that the incidence of this phenomenon is about 0.7/1000 inhabitants/ year. **Sudden death** happens in **20-25%** of cases in **apparently healthy individuals**, as a first manifestation of an unacknowledged underlying pathology. The 5-10% of sudden death cases occur in absence of evident structural cardiac anomalies in structurally normal hearts (*sine materia* sudden death), in presence of electrophysiological disorder which determines an electric instability responsible of the onset of ventricular arrhythmias, same as the case of long QT syndrome (LQTS), the Brugada syndrome (BS), catecholaminergic polymorphic ventricular tachycardia (CPVT). In a study conducted by British Steering Group, in 32 consecutive cases of sudden arrhythmic death sine materia, not selected by age, the cardiac screening of first-degree relatives has revealed the presence of an inherited heart disease in 22% of examined families, and approximately half of the cases were LQTS.

In a Tan et al. analysis conducted on 43 families in which there was at least a case of sudden death under 40 years of age, in 40% of cases cardiac screening allowed to identify an inherited heart disease (LQTS, CPVT, BS and arrhythmogenic right ventricular cardiomyopathy ARVC). A more recent study conducted in England on 262 relatives (of which 70% of first grade) of 57 families with at least one sudden death case, has documented an inherited heart disease in 53% of the examined families: 17% of cases were about a structural heart disease (ARVC, hypertrophic cardiomyopathy HCM, dilated cardiomyopathy, left ventricular noncompaction). In the remaining 26% the diagnosis was LQTS or BS.

Cardioscreen – Prevention of sudden cardiac arrest test

Cardioscreen – Prevention of sudden cardiac arrest is adiagnostic test, developed by GENOMA Group, which allows us to perform a multiple genetic analysis **to evaluate the presence of mutations associated with sudden cardiac death *sine materia***. Therefore, the test, allows us to identify patients at genetic risk of potentially mortal cardiac events through the analysis of their DNA.

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Whom is CardioScreen-Prevention sudden cardiac arrest test for?

Genetic screening test for predisposition to sudden cardiac arrest is particularly recommended for those who knows about a case of sudden cardiac death in their family (included sudden infant death), heart failure or transplant, which suggest inherited cardiac pathological substratum.It is useful to inspect also the relatives of accidental death victims caused by sudden illness, for example during the driving of a vehicle, to evaluate whether the event is attributable to a syncopal episode or to a sudden cardiac death.

The analysis of the family tree crossed with genetic screening, can provide information about the modality of transmission of inherited heart disease and its level of penetrance in any family members. Genetic screening of mutations associated to inherited cardiomyopathies is useful to arrange prevention strategies so that unexpected serious events don't occur and don't affect members of the same family.

Furthermore is particularly useful as a prevention instrument in case of:

- Professional or amateur agonistic activity, also for individuals with no familiarity
- Young individuals (younger than 40 years) with idiopathic cardiac symptomatology
- Children and teenagers with a suspect clinical picture for QT anomalies or cardiac rhythm

The geneticist, as mutually agreedupon with the cardiologist, upon informed agreement of person concerned, will suggest whether to proceed or not with the genetic screening.

What are CardioScreen- Prevention sudden cardiac arrest test benefits?

The possibility to identify an at-risk individual for inherited cardiomyopathies or for sudden cardiac arrest, represents today the best method to express an early diagnosis of a potential pathology, and, therefore, to reduce mortality and related morbidity. Members of inherited high-risk families, and in particular who is affected by an idiopathic cardiac symptomatology, can ask for a genetic consultation and discuss the own genetic-clinical situation with the geneticist.

This evaluation will be able to promote the genetic test to verify if the patient is carrier of a mutation associated to an inherited cardiomyopathy and sudden cardiac arrest. If the test is positive, the examination will be extended to patient's relatives to identify at-risk individuals of the nuclear family.

The information obtained from the genetic test can generate remarkable **benefits**, such us:

1. The identification of family members at **high risk of inherited cardiomyopathy**;

2. The organisation of an adequate **medical examination program** reserved for high risk individuals so to facilitate the adoption of **the most effective preventive measures** (for example implantable defibrillators or antiarrhythmic pharmacologic therapies);

3. The knowing of the possibility of **transmission of genetic mutation** to the progeny and the identification of individuals children with germinal genic mutations at high risk.

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How is CardioScreen- Prevention sudden cardiac arrest test done?

CardioScreen test is done through the taking of a haematic sample. By means of a complex laboratory analysis, the DNA is isolated from nucleated cells and amplified by C-reactive protein (CRP) technique. Later, thanks to an innovative technological process of massive parallel sequencing (MPS), which employs Next Generation Sequencing (NGS) techniques using ILLUMINA sequencers, they completely sequence at an elevated in – depth reading, 160 genes (exons and adjacent intragenic regions, ± 5 nucleotides) connected to inherited cardiac conditions and correlated to sudden cardiac arrest (Table1).

Genetic sequences obtained are analysed through an advanced bioinformatics analysis to determine the presence of potential mutations on genes taken under exam.

Achievable result with CardioScreen-Prevention sudden cardiac arrest test

"POSITIVE"-Presence of one or more mutations: it indicates the test has revealed one or more mutations of one (or more) genes related to inherited cardiomyopathies. Our geneticist, during genetic counselling, will explain in a detailed way the meaning of the test result.A positive result doesn't mean that the patient to whom the mutation has been found will have a serious pathological cardiac event, but it means that the patient has a mutation related to inherited cardiomyopathies, that is a **greater risk** compared with a person who doesn't have that specific mutation. In a suspect situation, the test is useful for confirming diagnostic hypothesis that has to be verified. As a matter of fact, not all people who carry a mutation undergo to gravely pathological heart events; although these mutations considerably increase the risk that a sudden cardiac event may occur during life or during cardiac stress like a sports performance. The identification of a predisposing mutation allows to establish a protocol of clinical controls and to evaluate the opportunity of preventive interventions like implantable defibrillators or antiarrhythmic drug therapies. The positive test result also allows you to extend screening to other family members at risk who wish to do it. In the latter, the analysis has a predictive value, because allows distinguishing, within these families, carriers of potentially dangerous mutations from noncarriers, by precisely identifying high-risk individuals and those whose risk is comparable to that of the general population. In this way, the first can be started in a targeted manner to specific surveillance or prophylactic programs, while the seconds can be directed to the controls planned for the general population.

Mutations observed through **CardioScreen-Prevention sudden cardiac arrest** test can be included in the following prognostic categories:

- **Known pathological meaning**;
- **Benign meaning,** since they can be found in normal individuals and they are pathologically meaningless
- **Pathological uncertain meaning**, since they aren't known or characterized from the scientific-medical community. In this case, further investigation can be necessary to clarify the variation meaning.

"NEGATIVE" - Absence of mutations: it indicates the test didn't detect the presence of mutations in the analysed genes. However is important to underline that a negative result doesn't mean the patient has zero

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risk to meet with a potentially serious cardiac event or to develop a cardiomyopathy in the own lifetime. The risk for this people is the same as for the general population, this because not all forms of cardiomyopathy and sudden cardiac arrest has to be connected to genetic causes.

CLINICAL RECORDS IN LITERATURE

In a study conducted on 100 consecutive cases of sudden deaths in young people (up to 40 years), occurring in the Lazio Region between 2001 and 2005, the autopsy allowed to identify coronary artery disease in 30% of cases, (mostly atherosclerotic) and cardiomyopathy in 22% of cases. Among cardiomyopathies, the most recurring is the arrhythmogenic right ventricular cardiomyopathy (AVRC, 12%) followed by hypertrophic cardiomyopathy (HCM, 4%). Myocarditis was observed in 2% of cases and mitral valve prolapse in 3%. Finally, in 20% of cases the heart resulted structurally normal during autopsy while in the remaining 28% the causes of death were not closely related to heart disease.

There is an English perspectival study conducted by Behr et al. in England on adult subjects, that is focused only on unexpected cardiac death with negative autopsy (defined sudden arrhythmic death syndrome SADS) some of which can be attributed to inherited arrhythmic syndromes. In this study was observed an SADS annual incidence of 0.16 cases on 100 000 per year (500 cases per year), with a predominance in young man. In absence of structural heart disease, the pathophysiological substratum of sudden death is represented by inherited primitive electrophysiological disorders, represented by ion channels diseases, mainly LQTS, BS and CPVT. The study also identify a 18% prevalence of a positive family history for other cases of sudden or unexplained accidental deaths, suggesting the possibility of an underlying inherited heart cause.

In SADS study conducted by British Steering Group, in 32 consecutive cases of sudden arrhythmic death sine materia, not selected by age, cardiac screening of first-degree relatives has revealed the presence of an inherited heart disease in the 22% of examined families; approximately half of the cases were about LQTS.

Parameters employed to report genetic variants

The analysis is focused exclusively to genes listed in Table 1. It will be reported only the mutations classified as “known pathogenetics meaning” or with “uncertain meaning”, based on the scientific literature data and the classification included in the reference database Human Gene Mutation Database (HGMD), updated to the date of the sample. Furthermore, following the indications of American College of Medical Genetics (ACMG), it has been considered as pathogenetics or alleged pathogenetics, only the mutations with a value of Minor Allele Frequency (MAF) <5% (1000 Genomes Project), that can be attributed as the frequency of recurrence of the less common allele within the population.

Target Coverage

For Target Coverage it is meant the average number of readings obtained from the sequencing for each nucleotide base that constitutes the gene. The variants with a reading depth (number of reads) lower than 30X, are not highlighted by the bioinformatics analysis algorithm.

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Accuracy of CardioScreen- Prevention sudden cardiac arrest test

Current DNA sequencing techniques produce results with an accuracy superior to 99%. Although this test is very accurate it is always necessary to consider the exam limits, shown below.

CardioScreen- Prevention sudden cardiac arrest test limits

This exam evaluates only genetic diseases and the genes listed in the Table 1.

The test does not highlight other genetic diseases or genes not specifically investigated.

Furthermore, the exam is not able to highlight:

- Mutations localised in intragenic regions over ± 5 nucleotides from breakpoints;
- Deletions, inversions or duplications superior than 20bp;
- Germinal line mosaicisms (that are mutations present only in gametes).

A **"NEGATIVE" result – Absence of mutations** for investigated genes do not exclude the possibility of being carrier of a mutation localised in a genome region that wasn't investigated during the exam.

It is possible that some areas of our own DNA can't be sequenced, or that have a lower coverage than the limits set by the GENOMA Group experts to ensure an accurate analysis of the variants. These regions will not be included in the analysis in case they do not pass qualitative standard requested.

In some cases, the genomic analysis result can reveal a DNA variation or mutation with a clinical meaning uncertain or determinable, on the basis of the current scientific-medical knowledge.

The interpretation of genetic variation is based on the most recent available knowledge at the time the analysis is done. This interpretation could change in the future with the acquisition of new scientific and medical information on genome structure and could influence on the same evaluation of variations.

Some pathologies can be caused or regulated by more than one variant in the DNA in one or more genes. Some of these variants may not be identified or validated by the scientific community yet, and therefore not reported as pathogenetics at the time of the analysis.

The intrinsic limit of NGS methodology used is the lack of coverage homogeneity for each analysed region. This limitation translates in the possibility, inherited in NGS methods, that specific mutations of selected genes may not have been detected by the test.

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List of the analysed genes and investigated genetic diseases

Table 1: CardioScreen-Prevention sudden cardiac arrest

	DISEASE NAME	PhenoMIM	GENE
1	Atrial fibrillation, familial, 12	614050	ABCC9
2	Sitosterolemia	210250	ABCG5
3	Sitosterolemia	210250	ABCG8
4	Myopathy, actin, congenital, with cores	161800	ACTA1
5	Aortic aneurysm, familial thoracic 6	611788	ACTA2
6	Atrial septal defect 5	612794	ACTC1
7	Cardiomyopathy, dilated, 1AA, with or without LVNC	612158	ACTN2
8	Long QT syndrome-11	611820	AKAP9
9	Alstrom syndrome	203800	ALMS1
10	Cardiac arrhythmia, ankyrin-B-related	600919	ANK2
11	Hyperchylomicronemia, late-onset	144650	APOA5
12	Hypercholesterolemia, due to ligand-defective apo B	144010	APOB
13	Hyperlipoproteinemia, type Ib	207750	APOC2
14	Lipoprotein glomerulopathy	611771	APOE
15	Cardiomyopathy, dilated, 1HH	613881	BAG3
16	Cardiofaciocutaneous syndrome	115150	BRAF
17	Brugada syndrome 3	611875	CACNA1C
18	Brugada syndrome 4	611876	CACNB2
19	Long QT syndrome 14	616247	CALM1
	Ventricular tachycardia, catecholaminergic polymorphic, 4	614916	
20	Cardiomyopathy, hypertrophic, 19	613875	CALR3
21	Ventricular tachycardia, catecholaminergic polymorphic, 2	611938	CASQ2
22	Cardiomyopathy, familial hypertrophic	192600	CAV3
	Long QT syndrome 9	611818	
23	Noonan syndrome-like disorder with or without juvenile myelomonocytic leukemia	613563	CBL
24	Homocystinuria, B6-responsive and nonresponsive types	236200	CBS
25	Hyperalphalipoproteinemia	143470	CETP
26	Ehlers-Danlos syndrome, type III	130020	COL3A1
27	Ehlers-Danlos syndrome, classic type	130000	COL5A1
28	Ehlers-Danlos syndrome, classic type	130000	COL5A2
29	Cardioencephalomyopathy, fatal infantile, due to cytochrome c oxidase deficiency 2	615119	COX15
30	Atrioventricular septal defect, partial, with heterotaxy syndrome	606217	CRELD1
31	Cardiomyopathy, dilated, 1II	615184	CRYAB

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